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Nitric oxide pathway in rectoanal inhibitory reflex of opossum internal anal sphincter

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Abstract

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The role of nitric oxide in relaxation of the internal anal sphincter (IAS) in response to the rectoanal reflex was studied in the opossum. Resting pressures in the IAS (IASP) were monitored using low-compliance continuously perfused catheters. The NO-synthase inhibitor L-NG-nitro-arginine (L-NNA) caused significant and dose-dependent suppression of the decrease in IASP in response to the reflex mimicked by the rectal balloon distention. NO-synthase inhibitor blocked IAS relaxation in response not only to rectoanal reflex but also to other neural stimuli such as sacral nerve stimulation, local intramural stimulation, and the nicotinic ganglionic stimulant 1,1-dimethyl-4-phenylpiperazinium. Suppression of the neurally mediated IAS relaxation by L-NNA was stereoselective; D-NNA had no effect on the relaxation. The suppression of the rectoanal reflex-induced IAS relaxation by L-NNA was completely reversed by NO precursor L-arginine stereoselectively as D-arginine failed to reverse the suppressed IAS relaxation. Sodium nitroprusside caused a decrease in IASP that was modified neither by the neurotoxin tetrodotoxin nor by L-NNA. Furthermore, the decrease in IASP by the direct-acting beta-adrenoceptor agonist isoproterenol was also not modified by the inhibitor of NO synthase. It is concluded that NO or an NO-like substance is an important mediator of IAS relaxation in response to noradrenergic, noncholinergic nerve stimulation.
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Publishing and Reprint Information

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